REVIEW ARTICLE

Sports Med 2003; 33 (7): 517-538
0112-1642/03/0007-0517/$30.00/0

Heart Rate Monitoring

Applications and Limitations

Juul Achten and *Asker E. Jeukendrup*

Human Performance Laboratory, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, United Kingdom

Contents

Abstract

Over the last 20 years, heart rate monitors (HRMs) have become a widely used training aid for a variety of sports. The development of new HRMs has also evolved rapidly during the last two decades. In addition to heart rate (HR) responses to exercise, research has recently focused more on heart rate variability (HRV). Increased HRV has been associated with lower mortality rate and is affected by both age and sex. During graded exercise, the majority of studies show

that HRV decreases progressively up to moderate intensities, after which it stabilises. There is abundant evidence from cross-sectional studies that trained individuals have higher HRV than untrained individuals. The results from longitudinal studies are equivocal, with some showing increased HRV after training but an equal number of studies showing no differences. The duration of the training programmes might be one of the factors responsible for the versatility of the results.

HRMs are mainly used to determine the exercise intensity of a training session or race. Compared with other indications of exercise intensity, HR is easy to monitor, is relatively cheap and can be used in most situations. In addition, HR and HRV could potentially play a role in the prevention and detection of overtraining. The effects of overreaching on submaximal HR are controversial, with some studies showing decreased rates and others no difference. Maximal HR appears to be decreased in almost all 'overreaching' studies. So far, only few studies have investigated HRV changes after a period of intensified training and no firm conclusions can be drawn from these results.

The relationship between HR and oxygen uptake ($\dot{V}O_2$) has been used to predict maximal oxygen uptake ($\dot{V}O_{2max}$). This method relies upon several assumptions and it has been shown that the results can deviate up to 20% from the true value. The HR-$\dot{V}O_2$ relationship is also used to estimate energy expenditure during field conditions. There appears to be general consensus that this method provides a satisfactory estimate of energy expenditure on a group level, but is not very accurate for individual estimations.

The relationship between HR and other parameters used to predict and monitor an individual's training status can be influenced by numerous factors. There appears to be a small day-to-day variability in HR and a steady increase during exercise has been observed in most studies. Furthermore, factors such as dehydration and ambient temperature can have a profound effect on the HR-$\dot{V}O_2$ relationship.

Heart rate monitors (HRMs) have become a common training tool in endurance sports. Most endurance athletes have at least tried HRMs and many use them consistently to monitor their training and to help them train at the planned intensity. HRMs have developed rapidly from large instruments suitable only for laboratory use (around the 1900s) to the size of a watch in recent years. There have been developments in the accuracy of the measurements, increased storage capacity, and new functions have been added. There are various ways in which HRMs can be used and this review discusses their possible applications.

In addition to the applications mentioned in this article, heart rate (HR) monitoring also has various limitations. The relationship between HR and other physiological parameters (such as oxygen uptake [$\dot{V}O_2$] or blood lactate concentration) is often determined in an exercise laboratory. Some factors have been identified that can potentially influence these relationships. The most important factors are discussed in this review.

1. The Development of Heart Rate Monitoring (HRM)

1.1 History of HRM

For several centuries, HR monitoring consisted of placing an ear on the patients' chest. 200 years ago the stethoscope was invented by Rene Laennec which made it possible to listen more accurately to the heart beat. However, it was still not possible to create an accurate picture of the changes that occur within the heart or to monitor HR during exercise. At the start of the 20th century, the Dutch physiologist Willem Einthoven developed the first electrocardiograph (ECG). With an ECG it is possible to make a graphic recording of the electric activity, which is present in the heart. The ECG is composed of three sections, a P wave, a QRS wave and a T wave. These waves represent the depolarisation of the atria, depolarisation of the ventricles and repolarisation of the ventricles, respectively.

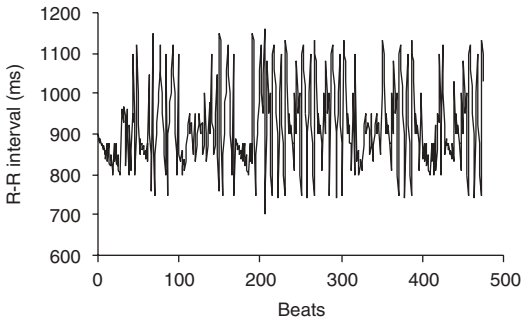
Soon after the invention of the ECG, the Holter-monitor was developed. The Holter-monitor is a portable ECG capable of making a continuous tape recording of an individual's ECG for 24 hours.[1] However, the relatively large control box and the wires necessary to record the changes in the electric field created by the heart, make the Holter-monitor unsuitable for recording HR during exercise in all conditions.

In the 1980s, the first wireless HRM was developed, consisting of a transmitter and a receiver. The transmitter could be attached to the chest using either disposable electrodes or an elastic electrode belt. The receiver was a watch-like monitor worn on the wrist.[2] The development of this relatively small wireless monitor resulted in an increased utilisation of HRMs by athletes. As a consequence, the objective measure of HR replaced the more subjective perceived exertion as an indicator of exercise intensity. In the 20 years after the development of the first monitor, HRMs have been developed with larger memory capacity. This allows for storage of HR data from more exercise sessions. HR data can be 'downloaded' into a computer, which makes the analysis of a training, race or exercise test possible. More recently, HRMs have been equipped with a calorie-counting feature and estimations of maximal oxygen uptake ($\dot{V}O_{2max}$). Another relatively recent development in HR monitoring is the measurement of heart rate variability (HRV) that may have various applications. These features and their reliability and validity will be discussed in the following sections.

1.2 Heart Rate Variability (HRV)

Even when HR is relatively stable, the time between two beats (R-R) can differ substantially. The variation in time between beats is being defined as HRV. Currently, variations in inter-beat intervals are used as an index of autonomic responsiveness. As will be explained in section 1.2.2, high HRV is associated with high $\dot{V}O_{2max}$ values while it has been found that low HRV is associated with increased mortality,[3] the incidence of new cardiac events[4] and risk of sudden cardiac death in asymptomatic patients.[5]

HRV is assessed by examining the beat-to-beat variations in normal R-R intervals. Originally, HRV was quantified in a time-domain, i.e. R-R intervals in milliseconds (ms) plotted against time (figure 1). The standard deviation of the R-R intervals (SDNN), that is the square root of variance, can show short-term as well as long-term R-R interval variations. Differences between successive R-R intervals provide an index of cardiac vagal control. This can be quantified by calculating the root mean square successive difference (r-MSSD) of all R-R intervals and the number of adjacent R-R intervals differing more than 50ms expressed as a percentage of all intervals over the collection period (pNN50). In figure 2, an ECG of an individual at rest is



R-R intervals in time-domain	
AverageNN (ms)	Average of all normal R-R intervals
SDNN (ms)	Standard deviation of all normal R-R intervals
r-MSSD (ms)	Root mean square successive difference
pNN-50 index (%)	Percentage of differences between adjacent normal R-R intervals that are >50ms

Fig. 1. Example of R-R interval time between each subsequent beat measured over a 7-minute period at rest (~500 beats) and common ways to express heart rate variability in the time-domain.

displayed. For clarity of the example, the ECG only consists of 11 beats, it should be noted that the calculations normally are performed over a longer period. In figure 2, both the R-R interval time and the difference between each two adjacent R-R intervals are presented in ms. The average R-R interval in this example is 925ms with a SDNN of 40ms. To calculate r-MSSD, the differences between adjacent intervals are squared and the mean is calculated. The squared root of the calculated mean, 62.6ms, is the r-MSSD. Of the nine calculated differences, six appear to be larger than 50ms, giving a pNN50-index of 67%.

In contrast to the time-domain measures of HRV, recent developments in microprocessor technology have enabled the calculation of frequency measures based on mathematical manipulations performed on the same ECG-derived data. Instead of plotting the HRV as the change in R-R intervals over time, it is plotted as the frequency at which the length of the R-R interval changes. In figure 3, the power spectrum of the R-R intervals is depicted. The main parameters on the frequency domain are very low frequency power (VLFP), low frequency power (LFP), high frequency power (HFP), ratio between LFP and HFP (LFP/HFP) and total power (TP). The measurements at different frequencies are usually expressed in absolute values of power (milliseconds squared). In case the data of the different power components are not normally distributed, the data are often log-transformed. HFP and LFP may also be measured in normalised units, which represent the relative value of each power component in proportion to the TP minus the VLFP component.

The peaks at different frequencies reflect the different influences of the parasympathetic and sympathetic nervous system.[6-11] Part of the HRV is caused by respiratory sinus arrhythmia. During inspiration the R-R interval will decrease, while the opposite is seen during expiration. Respiratory sinus arrhythmia is mainly mediated by parasympathetic activity to the heart,[12] which is high during expiration and absent or attenuated during inspiration. It has been shown in both clinical and experimental settings that parasympathetic activity is a major contributor to the HFP component of the power spectrum.[6,7,9,11] The evidence for the interpretation of the LFP component is much more controversial. The LFP is seen as a marker of sympathetic modulation by some authors.[8-10] while others suggest it is a parameter that includes both sympathetic and para-

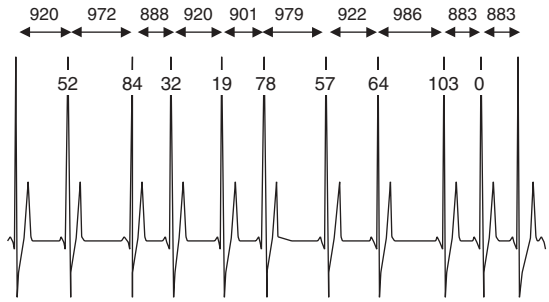
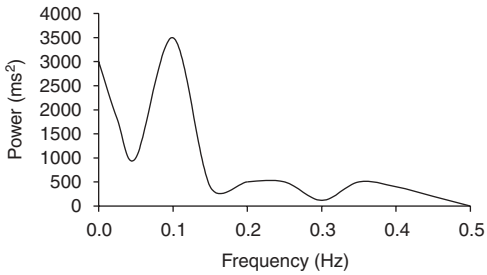


Fig. 2. Example of an ECG output over 11 beats. R-R interval times and difference between adjacent R-R intervals are displayed.



R-R intervals in frequency-domain	
Total power (ms^2)	The power in the heart rate power spectrum between 0.00066 and 0.34Hz
VLFP (ms^2)	The power in the heart rate power spectrum between 0.0033 and 0.04Hz
LFP (ms^2)	The power in the heart rate power spectrum between 0.04 and 0.15Hz
HFP (ms^2)	The power in the heart rate power spectrum between 0.15 and 0.36Hz
LFP : HFP ratio	

Fig. 3. An example of the power spectrum which shows the magnitude of the variability as a function of frequency. The most commonly found areas in the power spectrum, which represent different influences of sympathetic and parasympathetic nervous system, are displayed in the box. **HFP** = high frequency power; **LFP** = low frequency power; **VLFP** = very low frequency power.

sympathetic influences.[6,7,13] The ratio of LFP to HFP is considered to reflect the sympatho-vagal balance and high values suggest a sympathetic predominance.[11,13,14] It has been shown that pNN50 and r-MSSD will provide the same information as the HFP component when calculated from both short-term[15] and long-term recordings.[16]

Both age and sex appear to be important determinants of HRV in healthy individuals. Jensen-Urstadt et al.[17] studied 102 men and women varying in age between 20 and 70 years. ECGs were collected for 24 hours for each individual and both time and frequency domain variables of HRV were calculated. It was shown that there was a strong negative correlation between TP, VLFP, LFP, HFP and SDNN, r-MSSD and pNN50-index and age. In the 60–69 year group, TP was ~30% lower than in the 20–29 year group. Overall these results suggest that HRV decreases with increasing age. Similar results have been reported by others.[18-21] In the study by Jensen-Urstadt et al.,[17] women had lower TP, VLF, LFP, LF/HF ratio, and SDNN than men. These data confirm the findings of other studies,[18,19] showing that women have lower HRV than men.

When using power spectral analysis to interpret R-R interval data, several confounding factors should be considered. As already mentioned, part of the HRV is determined by respiratory sinus arrhythmia and it is therefore logical that any change in breathing pattern, will have an influence on the power spectrum. Brown et al.[22] studied the power spectrum of nine healthy individuals, breathing at seven different frequencies at two different tidal volumes. They concluded that both respiratory rate as well tidal volume strongly influenced TP, LFP and HFP. TP was highest at low breathing frequencies (6–10 breaths/min) and it decreased when the frequency was increased above 10 breaths/min.

Another factor which can influence the power spectrum of HRV is body position. It has been shown that both at rest[11,23] and during exercise,[24] the power spectrum is significantly different when individuals are supine or upright. There is general agreement that HR is lower in a supine compared with an upright position, which can be ascribed to a higher vagal tone, leading to an increased TP in the power spectrum of HRV.[11,23] When individuals move from a supine to an upright position, blood will pool into the lower extremities, causing a drop in blood pressure. The change in blood pressure is picked up in baroreceptors located in the carotid sinus in the walls of the aortic arch and this will result in reflex tachycardia. It has been suggested that this baroreflex is mainly vagally mediated, and changes in baroreflex sensitivity are hence thought to be connected with changes in parasympathetic activity.[25]

1.2.1 Effects of Exercise on HRV

The effects of exercise on indices of HRV have been investigated on numerous occasions.[26] At the transition from rest to exercise, a decrease is seen in

SDNN[27-30] and in TP, HFP and LFP, expressed in absolute[27,29,31,32] and log transformed values,[8,30,32-35] indicating that the influence of the parasympathetic nervous system is decreased. In normalised units, HFP decreases[28,29,35] while LFP does not change at the start of exercise in the majority of studies.[27,28,32] No change has been observed in the ratio between LFP and HFP at the transition from rest to exercise in some studies,[30,33,34] although others reported an increased ratio.[28,29,31]

A few studies have investigated the influence of exercise intensity on HRV during exercise. In these studies, time and frequency domain variables have been calculated when individuals were performing graded exercise tests to exhaustion. When frequency power is expressed in absolute values, HRV tends to decrease progressively at intensities of up to 50% $\dot{V}O_{2max}$, while at higher exercise intensities, the values tend to level off. In 1995, Casadei and colleagues[27] studied HRV in 11 healthy men during a graded exercise test to exhaustion. HRV parameters were calculated at 43, 57, 72 and 86% $\dot{V}O_{2max}$. TP decreased from 602 ms^2 during the first stage, to 270, 207 and 158 ms^2 on the subsequent stages, respectively. HFP and LFP followed a similar pattern decreasing from 202 and 260 ms^2 to 131, 132, 128 and 77, 9, 0 ms^2 respectively. However, when the data are expressed in normalised units the study results are not conclusive. In two studies,[14,32] the HFP showed a slight decrease at intensities above 60% $\dot{V}O_{2max}$, while in another study the HFP increased considerably at near maximal exercise.[27] The LFP has been reported to progressively decrease with increasing intensity above ~30% $\dot{V}O_{2max}$ in one study,[32] while the onset of the decrease was at ~60% $\dot{V}O_{2max}$ in another study.[27] It is unlikely that methodological differences can explain the different findings because the type of subjects and the exercise protocol used in both studies were very similar.

1.2.2 Effects of Exercise Training on HRV

It has been known for a long time, that trained endurance athletes have profound bradycardia. The underlying mechanisms for this decreased resting HR have been extensively investigated and numerous possible causes have been proposed. Part of the decrease is due to a decrease in intrinsic HR, i.e. the HR obtained with complete removal of autonomic influences, which is presumably related to membrane stabilisation of the conduction system cells.[36-38] Enhanced vagal tone to the sinus node has also been proposed to play a role in sinus bradycardia,[39-43] whether the sympathetic nervous system also contributes to the lower resting HR is still controversial.[44-47] The latter two factors (vagal and sympathetic influences) are reflected in the different measures of HRV.

The differences in HRV between trained and untrained individuals have also been investigated on numerous occasions. When looking at the time-domain variables, in most studies trained individuals had significantly higher R-R interval times,[15,48-55] SDNN,[15,48-50,53-56] pNN50-index[15,48,51,52] and r-MSSD[48,51,56] compared with their age- and weight-matched sedentary controls. Three studies[28,51,52] failed to find significant differences in SDNN between trained and untrained individuals and differences in rMSSD were not detected.[15] When the data are interpreted using frequency domain variables, the results are slightly less consistent. A difficulty with the comparison between these studies is the representation of the data. While some investigators reported absolute values, others used normalised units or log-transformed data. In the studies that provided absolute power data, TP, HFP and LFP were similar[15,48,53] or significantly higher[49,50,54] in athletes compared with sedentary individuals in most studies, while only one study[55,57] showed the opposite. The HFP expressed in normalised units was significantly higher in the trained individuals compared with sedentary individuals in four out of

six studies.[28,53,56,58] Puig et al.[54] did not find any differences in the HFP or LFP. Although Shin et al.[58] and Macor et al.[53] found differences in the HFP component, they were unable to show any differences in LFP. On the other hand, the trained individuals in the study of Dixon et al.[28] and Janssen et al.[52] had significantly lower LFP compared with their sedentary counterparts. In summary, both the time-domain variables and the HFP variable generally appear to be higher in trained individuals compared with sedentary individuals, indicating that HRV is higher in trained individuals. There seems to be less consistency in the results regarding the LFP component. It is difficult to attribute this diversity in results to the differences in study design, since most studies reviewed have used similar study participants and test protocols.

Although it would now be easy to conclude from the above-mentioned studies that training increases HRV, studies are needed to investigate the direct effect of training on indices of HRV. Over the last 5 years, several studies have addressed this question; however, the results of these studies are inconsistent. While some studies found an increased SDNN,[59-62] HFP[63-66] or LFP[60,63,64] after training, others could not detect any differences in these variables.[57,60,64,65,67-69] It could be argued that the differences found between these studies are due to methodological differences.

The studies described above have used a variety of study participants, training protocols and data representation. However, the main factor which could have influenced the study results is the duration of the training programme followed by the experimental individuals. Amano et al.[64] attempted to directly determine whether training duration has an effect on HRV by testing their subjects after 5 and 12 weeks of training. They reported no significant change after 5 weeks, but significant increases in TP, HFP and LFP after 12 weeks. The relatively short training duration of 6 weeks in a study by Boutcher et al.[67] also did not induce any changes. Studies using a training duration between 12 and 16 weeks induced significant increases in the HFP component[64-66] and training programmes between 26 and 39 weeks increased SDNN.[59-62] Only one study using a relatively large number of training weeks (20 weeks) was unable to show changes in any of the HRV variables.[68] Since most of the studies have only measured few HRV variables, it is difficult to draw firm conclusions from these data. However, it seems that long-duration training programmes show more favourable results than short-duration programmes.

In an effort to determine whether training intensity would affect HRV, Loimaala et al.[68] trained two groups of middle-aged men for 20 weeks. One of the groups performed exercise at 55% $\dot{V}O_{2max}$, while the other one performed exercise at 75% $\dot{V}O_{2max}$. Both groups trained on average four times per week for a duration of 33 minutes per session. After 20 weeks, no differences were found in any of the time and frequency domain variables of HRV in either intensity group.

While most cross-sectional studies show that endurance-trained individuals have higher HRV than their age- and weight-matched controls, the results from longitudinal studies are less conclusive. The data suggest that the duration of the exercise programme might be an important factor when looking at the effects of exercise training on HRV. However, the fact that endurance-trained individuals have consistently higher HRV than untrained individuals suggests that vigorous training programmes are necessary to induce changes in HRV and that in addition to exercise duration, exercise intensity and training volume may also play a role.

HRV analysis has been proven to be a simple non-invasive technique that evaluates the autonomic modulation of HR through measurements of instantaneous beat to beat variations in R-R interval length. Furthermore, it is an easy tool to non-inva-

sively explore the sympathovagal interaction in different conditions.[70]

1.3 Accuracy

The accuracy of the wireless HRMs has been extensively investigated. In 1988, Léger and Thivierge[71] tested 13 different HRMs during rest, exercise and recovery. It was concluded from this study that only four of the HRMs were valid when compared with an ECG. All four HRMs were based on the principle of the chest electrode. The HRMs which scored low on validity and reliability, used electrodes placed on fingers or hands or used photocells at the earlobe.[71,72] Macfarlane et al.[73] compared seven HRMs with an ECG in the same year and reached a similar conclusion. The HRMs using chest electrodes (Polar Sport Tester, Monark Trim guide 2000 Chest and Exersentry) produced both a mean bias and variability of less than 1.0 beat/min throughout their functional range, while monitors using different techniques produced HR data which was very deviant from the ECG data. The correlation coefficient of 0.9979 obtained by Seaward et al.[74] when data of a portable HRM was compared with an ECG, reflected the precision and accuracy of the HRM. Furthermore, in 1991 Godsen et al.[75] compared HR data collected with a wireless HRM with HR data collected by an ECG. The conclusion of the study was that the HRM was within 6 beats/min of the actual HR 95% of the time. In the studies by Godsen et al.[75] and Seaward et al.[74] the HRMs were validated during rest and exercise at different intensities. Recently, Goodie et al.[76] validated the wireless HRM during mental stress. The average HR of 30 individuals during a mental stress test was 80.7 ± 10.4 when measured using an ECG and 81.3 ± 10.4 with a wireless HRM ($r = 0.980$, $p < 0.0001$). The accuracy in the determination of the R-R interval was also investigated. Kinnunen and Heikkila[77] showed that in 95.4% of the R-R intervals, the difference between the Polar Vantage NV and the Polar R-R recorder was within 1ms. Ruha et al.[78] reported that Polar R-R recorder was both reliable and valid when tested against an ECG.

Therefore, HRMs using chest electrodes are considered to be both valid and reliable during physically and mentally stressful conditions. In addition, the measurement of HRV with HRMs has also been shown to be valid and reliable.

2. Main Applications of HRM

2.1 Monitoring Exercise Intensity

All training programmes consist of three key components: frequency of exercise sessions, duration of each session and exercise intensity. The sum of these training components has been previously described as the training impulse.[79] A training impulse is intended to result in a positive training adaptation and improved performance. However, it is known that excessive training impulses can result in deteriorated performance and ultimately in the development of overtraining syndrome.[80-82] Often there is a fine line between the optimal training impulse and a training impulse that will deteriorate performance. Therefore, it is believed that it is important to carefully monitor all three components of a training programme. The duration and frequency components of a training programme are relatively easy to monitor and there are several methods available to measure the intensity component.[83] When determining the best possible way to monitor exercise intensity, a balance has to be found between validity of the parameter and practicality of using that parameter for intensity measurements. Exercise intensity is usually defined as the amount of energy expended per minute to perform a certain task (kJ/min).[83] The methods that are currently available to measure energy expenditure (EE) directly, can not (or only on rare occasions) be used in non-laboratory settings.

There are several methods that can be used to determine exercise intensity in the field. Speed, for instance, can be used to accurately monitor the intensity of exercise in some modes of exercise, such as swimming and to some extent running. However, in sports such as cycling and cross-country skiing, speed will not always reflect the intensity. In these sports, the speed-intensity relationship can be affected by factors such as surface, undulating terrain and ambient conditions.[83]

It was established some time ago that HR and $\dot{V}O_2$ (i.e. EE) are linearly related over a wide range of submaximal intensities.[84] By determining the relationship between HR and $\dot{V}O_2$, HR can then be utilised to estimate $\dot{V}O_2$, which will give a fair reflection of the intensity of work that is being performed. With the development of the portable, wireless HRMs, HR has become the most commonly used method to get an indication of the exercise intensity in the field. HR is easy to monitor and shows a very stable pattern during exercise and athletes can immediately use the HR data to adjust the intensity of a work bout if necessary.

In a recent American College of Sports Medicine position stand on 'the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults'[85] a general classification of physical activity intensity was given using $\%HR_{reserve}$ ($HR_{max} - HR_{rest}$) and $\%HR_{max}$ to express intensity. Intensity was divided into six different categories ranging from very light to maximal. This classification makes it possible to estimate the intensity of an exercise bout expressed as $\%\dot{V}O_{2max}$ or metabolic equivalents, without determining the individual relationship between HR and $\dot{V}O_2$. It is important to note that the intensity obtained from this table will only give an indication of the true intensity and the individual relationship between HR and $\dot{V}O_2$ needs to be determined for a more accurate estimation.

Optimal use of HR as a measure of exercise intensity can be reached when the individual relationship between HR with more direct indicators of EE is established. By measuring $\dot{V}O_2$ and HR concurrently on a variety of intensities in a laboratory, HR can later be used to predict EE in the field if the exercise conditions are the same. In addition, the HR zones which coincide with the accumulation of lactic acid in the blood are often used to indicate various intensity zones.[86-88] In a quest for a non-invasive way to determine this HR zone, Conconi and colleagues[89] proposed a method to determine the anaerobic threshold based only on HR. Their results showed the expected linear relationship between HR and running speed at submaximal speeds but a plateau in HR at high running speeds. They reported that the deflection point of the HR-running speed relationship occurred at the same time as the anaerobic threshold. Although Conconi et al[89] were able to show a levelling off of HR in all 210 runners tested, other researchers who attempted to repeat the study, only found a plateau in a certain percentage of the individuals.[90] Furthermore, numerous authors have reported that the HR deflection point overestimates the directly measured lactate threshold.[91-93]

Jeukendrup and colleagues[94] stated, after careful consideration of the literature available on the Conconi-test, that the occurrence of the HR deflection is an artefact rather than a true reflection of the lactate threshold. The main criticism lies in the fact that in the Conconi-protocol, stage duration decreases with increasing exercise intensity. It has been argued that when the duration of a running stage is very short, the adaptation of the circulatory system to a certain speed will be incomplete and HR will start to lag behind progressively.[94] In an attempt to correct for this apparent flaw in Conconi's test, an adapted protocol was developed in which stage duration was fixed (30 seconds per stage) rather than running distance.[95] However, by having stages as short as 30 seconds, higher speeds can be attained during

which it is less likely to obtain a steady-state HR, increasing the likelihood of detecting HR deflection.[96] So, even in the adapted Conconi-test, the occurrence of the HR deflection is not a physiological phenomenon, but rather an effect of the protocol used.

In summary, the most important application of HR monitoring is to evaluate the intensity of the exercise performed. The intensity of an exercise bout is a key factor in determining the effect of a training session. HR shows an almost linear relationship with $\dot{V}O_2$ at submaximal intensities and can therefore be used to accurately estimate the exercise intensity. However, it should be mentioned that the relationship between HR and $\dot{V}O_2$ is individual and for precise estimations of exercise intensity, the relationship should be determined for each individual.

2.2 Detecting/Preventing Overtraining

2.2.1 Background Overtraining

Overtraining in athletes results from long-term stress or exhaustion due to prolonged imbalance between training in combination with other external and internal stressors and recovery.[97-99] The cardinal symptom of overtraining, or its less serious counterpart overreaching, is decreased performance.[80,88] Some of the additional symptoms are early fatigue, changes in mood state, muscle soreness, and sleeping disorders. In 1958, Israel[100] defined overtraining according to the effects it has on the autonomic nervous system. He distinguished between a sympathetic and a parasympathetic form of overtraining. The latter, parasympathetic and probably more chronic form of overtraining is dominating in endurance athletes and leads to a relatively bad prognosis because it often requires prolonged recovery periods.[98] Due to the seriousness of the syndrome, it is important to detect it in an early stage. So far, no single marker of overtraining has been determined. However, HR and HRV are closely related to parasympathetic and sympathetic activity and changes in the autonomic nervous system due to overtraining may be reflected in changes in HR and HRV.

2.2.2 Changes in Heart Rate Associated with Overtraining

In most studies, no differences were found in resting HR between normal and overreached state.[80,81,88,98,101] However, some early studies reported increased resting HR in overtrained individuals.[100,102,103] In addition, sleeping HR appears to be increased when individuals are overreached.[80,104] It has been suggested that sleeping HR is a more reliable measure since it is less likely to be affected by confounding variables.[80,83]

In a study by Billat et al.,[105] it was shown that HR was decreased from 155 to 150 beats/min at 14 km/h in runners after a period of intensified training. More recently, Hedelin et al.[97] also found significantly decreased HR (approximately 5 beats/min lower) at five different submaximal intensities after 6 days of increased training load. Others found similar submaximal decreases in HR in their study participants after a period of intensified training.[82,101,106] However, Urhausen et al.,[98] Halson et al.[88] and Jeukendrup et al.[80] reported that their study participants in the overreached state had similar submaximal HR compared with normal conditions.

HR during maximal exercise has been shown to decrease when individuals are overreached. In 1988, Costill et al.[106] showed that after 10 days of intensified training, the average maximal HR in 12 male swimmers significantly decreased from 175 ± 3 to 169 ± 3 beats/min. Jeukendrup et al.[80] showed that after 14 days of intensified training, maximal HR of cyclists declined significantly from 175 ± 3 to 169 ± 3 beats/min. Similar results were found in other studies.[88,97,101,107] However, Billat et al.[105] overtrained eight runners for 4 weeks and found that the maximal HR was the same after normal training

compared with overtraining. Therefore, most studies involving HR responses in overreached athletes, have found marked decreases in maximal HR whilst the changes in HR during submaximal exercise are less clear. Sleeping HR has been shown to increase and this has been suggested as one of the indicators for overreaching. Although resting HR may also be affected (increased with overreaching) this measure is less reliable and can easily be disturbed by external influences.

2.2.3 Changes in Heart Hate Variability Associated with Overtraining

As described in section 2.2.1, it has been suggested that a disturbance in the autonomic nervous system accounts for some of the symptoms in overtrained athletes. Since HRV will also be affected by changes in the autonomic nervous system, it might indicate early stages of overreaching or overtraining.[13,99]

The information about changes in HRV due to overreaching is sparse. Hedelin et al.[97] found no differences in the frequency domain parameters after the study participants performed 6 days of intensive training. In a case study, the effects of overtraining on a young cross-country skier were described.[108] This athlete showed remarkably high HFP and TP at the time of overtraining syndrome, suggesting an increased parasympathetic activity. Uusitalo et al.[99] performed a study in which 15 endurance-trained females were divided in a training group and a control group. The purpose of the experimental training period was to overreach the individuals in the training group. Five of the nine individuals in the training group became overreached (i.e. their $\dot{V}O_{2max}$ and maximal treadmill performance decreased, they were unable to continue training and experienced changes in mood state) while the other individuals showed some (but not all) symptoms of overreaching. The main finding in this study was the increased LFP in the training group and no change in the control group. No changes were observed in any of the other parameters (time and frequency domain).[99] It should be mentioned that in this study no information was provided regarding the time between the last exercise bout and measurements of HRV. It has been shown by Furlan et al.[109] that LFP remains elevated for up to 24 hours after an exercise bout. It is therefore possible that the effects seen on LFP are the result of a previously performed exercise bout.

The effects of intensified training on both HR and HRV appear to be unclear at present mainly because the number of studies that have addressed this problem is relatively small and very few studies increased training in a controlled and systematical manner to provoke a state of overreaching. Carefully controlled studies are needed to investigate the effects of intensified training on HRV before we can conclude that HRV is an important indicator of early overtraining.

2.3 Estimation of Maximal Oxygen Uptake ($\dot{V}O_{2max}$) and Energy Expenditure

It has been known for a long time that both $\dot{V}O_2$ and HR increase linearly with increasing exercise intensity up to near maximal exercise. It has been suggested that an individual's aerobic fitness is reflected in the slope of an HR-$\dot{V}O_{2max}$ curve.[110] Endurance training will reduce HR both at rest and during submaximal exercise at a given $\dot{V}O_2$.[111-114] With similar $\dot{V}O_2$ at a certain work rate before and after training but a lower HR after training, the slope of the line will decrease.[111,114] However, it was reported by Londeree and Ames[115] that when both $\dot{V}O_2$ and HR are expressed as a percentage of their maximum, no differences can be detected in the slope of highly-trained, moderately-trained and untrained individuals.

2.3.1 Estimation of $\dot{V}O_{2max}$

Over the last 50–60 years, the relationship between HR and $\dot{V}O_{2max}$ has been used to estimate $\dot{V}O_{2max}$. In the 1950s, Astrand and Ryhming[116]

tested more than 300 men and women at different exercise intensities and measured HR, workload and $\dot{V}O_2$. The data of this study were used to come up with a nomogram to predict $\dot{V}O_{2max}$.[116] To use this method of $\dot{V}O_{2max}$ prediction, an individual is only required to exercise for 6 minutes on one exercise intensity while HR (and in some cases $\dot{V}O_2$) is measured. The information on HR together with the individuals weight and gender is used on the nomogram to estimate $\dot{V}O_2$.

Another frequently used test involves exercise at three different intensities. HR and $\dot{V}O_2$ data will be plotted and a straight line should be drawn through the points of the plot. By extrapolating the line until the assumed maximal HR (220 − age in years[84]) for the particular age group, an estimation of $\dot{V}O_{2max}$ can be obtained.[110]

The accuracy of predicting $\dot{V}O_{2max}$ from submaximal HR has limitations. Such a method is based on the premise that the relationship between HR and $\dot{V}O_2$ is linear over the entire range of work intensities. This is an oversimplification, since the relationship is curvilinear at very low intensities and towards maximal exercise.[117] In addition, the estimation of maximal HR will result in an error. It has been shown in several studies that the standard deviation of the prediction of maximal HR lies between 8–12 beats/min.[118,119] The day-to-day variability in HR measurements described in section 3.1 can also cause an over- or under-estimation of the actual $\dot{V}O_{2max}$.

It has been suggested that $\dot{V}O_{2max}$ predicted from submaximal HR is generally within 10–20% of the person's actual $\dot{V}O_{2max}$.[110] Despite this rather large percentage, the predictive tests can be suitable for measuring individuals who are not capable of performing a maximal effort test (i.e. the elderly, pregnant women). It should be noted, however, that the prediction methods are only validated in a population of healthy, non-pregnant, young to middle-aged individuals.

As mentioned in section 1.2 on HRV, the most recent HRMs are equipped with the ability to predict $\dot{V}O_2$. Aerobic fitness ($\dot{V}O_{2max}$ in ml/kg/min) is predicted from resting HR, HRV, sex, age, height, bodyweight and self-assessment of the level of long-term activity.[120] The accuracy of this application has been tested on several occasions. In one study, 11 individuals repeated the 'fitness test' on the HRM on eight consecutive days at three different times during the day. The average individual standard deviation of all test results was less than 8% from the individual mean value. When data were evaluated per timepoint, the standard deviations were smaller.[121] The validity of the test was assessed in 52 healthy men aged 20–60 years. The individuals performed an exercise test in a laboratory to measure maximal aerobic power and this was also determined using the HRM. The mean error in $\dot{V}O_{2max}$ prediction was 2.2%; when repeated after 8 weeks of training the error was only –0.7%.[121]

2.3.2 Estimation of Energy Expenditure

The relationship between HR and $\dot{V}O_2$ is not only used to predict $\dot{V}O_{2max}$. The estimation of EE can also be based on this relationship. There are several ways to estimate EE in humans, including filling out activity-level questionnaires, using pedometers/actometers, direct/indirect calorimetry and the doubly labelled water technique. Although the last technique appears to be the most accurate for the determination of the EE in free living humans,[122] the high costs and the inability to obtain an activity pattern does not make this method always ideal. These problems are overcome when EE is estimated from calorimetry. However, the major drawback with this method is the fact that the equipment necessary to do the measurements can interfere with the normal performance of the activities. Estimating EE from HR is relatively cheap and easy to perform and has therefore been investigated in numerous studies.[122-128]

To use HR for the estimation of EE, the individual relationship between HR and $\dot{V}O_2$ needs to be determined. Measurements of $\dot{V}O_2$ can then be used to calculate EE at several different HRs. The main limitation of the use of HR for measuring EE is the almost flat slope of the relationship at low expenditure levels.[127] At rest, slight movements can increase the HR, while EE (i.e. $\dot{V}O_2$) remains almost the same. In addition, the estimation of EE from HR is sport-specific. It has been well documented that type of activity and posture can influence the relationship between EE and HR and can therefore affect the estimation of EE from HR.[126,129]

There appears to be general consensus that while the HR method provides satisfactory estimates of average EE for a group, it is not necessarily accurate for individual study participants.[122,130,131] For example, Spurr et al.[122] compared 24-hour EE by calorimetry and with the HR method in 22 individuals. The maximum deviations of the values of EE between the two methods varied between +20 and –15%. However, when the data were compared using a paired t-test, no significant differences were observed.

During intermittent exercise, the HR-EE relationship may not be as accurate. HR responds relatively slowly to changes in work rate. Therefore, a sudden increase in work rate will not immediately result in the HR that would be observed at that exercise intensity after a 3–5 minute adaptation to the work rate had been allowed. Similarly, when the work rate is decreased, HR will remain elevated for some time and only gradually return to the HR observed during steady-state conditions at this lower work rate.

In summary, when HR is used to estimate $\dot{V}O_{2max}$ or EE, a linear relationship between HR and $\dot{V}O_2$ is assumed. Although this is true for a large range of intensities, during very low and very high intensities the relationship becomes non-linear. Furthermore, when quick changes are made from low to high intensities (and vice versa), the HR response lags behind. This will introduce a small error when HR is being used to predict EE or $\dot{V}O_{2max}$. It is therefore suggested that HR can only be used to estimate $\dot{V}O_{2max}$ and EE on group level.

3. Factors Influencing Heart Rate During Exercise

As described in section 2, the relationship between HR and other parameters ($\dot{V}O_2$ and lactate concentration) is used to predict, estimate and monitor an individual's fitness level. Often these relationships are determined in an environment where temperature and humidity of the ambient air will be controlled. Furthermore, individuals will attempt to enter a test under the best possible circumstances, having had enough sleep, carbohydrates and fluids the day(s) before. However, in the field, numerous factors can influence the relationship determined during a laboratory test and this may have implications for the interpretation of the data obtained of HRMs.

This section describes the natural variation which occurs in HR. In addition, the most important influential factors on the response of HR on exercise are described. Physiological, environmental and other factors are listed and there will be a short explanation of how the relationship is altered. In addition, an indication will be given about the magnitude of the changes.

3.1 Day-to-Day Variability in Heart Rate

Even with the best equipment available, HR measurements can only be used to monitor exercise intensity when the intra-individual differences are small. The day-to-day variability in the HR response to a certain exercise stimulus has been investigated on several occasions. In a study published in 1949, Taylor[132] examined the stability of individual differences of the physiological response to exercise. On two different occasions, 31 individuals were measured while walking and running. The intra-

individual variability of HR during submaximal exercise averaged 4.1%. The variability dropped to 1.6% during maximal exercise. Astrand and Saltin,[133] showed that the day-to-day variation in maximal HR was approximately 3 beats/min. When average HRs of ten different exercise intensities were compared on two separate days in 11 individuals, Brooke et al.[134] reported that the average test-retest correlation for HR was 0.872 ± 0.03. In a study by Becque et al.,[135] four individuals performed 20 submaximal tests at 50W and 10 tests at 125W and 55% maximal work rate. Of all the parameters measured (ventilatory rate, $\dot{V}O_2$, blood pressure and HR), HR showed the lowest coefficient of variance (average 1.6%), which was in close agreement with the results of Taylor.[132] The individuals in a study by Brisswalter and Legros[136] who had markedly higher $\dot{V}O_{2max}$ values than the individuals in Becque et al.'s study ($\dot{V}O_{2max}$ 71.2 ± 2.1 vs 58.1 ± 8.9 ml/min/kg, respectively), showed similar coefficients of variation of 1.6 ± 1.3% in HR over four tests.

From the above-mentioned studies, it has become clear that although the test-retest reliability of HR is high, a small day-to-day variation exists. Even under controlled conditions, changes of 2–4 beats/min are likely to occur when individuals are measured on different days. To minimise the effect of this day-to-day variability on the prediction of the work rate, HR zones are often prescribed to athletes rather than single HRs.

3.2 Physiological Factors

Several physiological factors influence the HR response to exercise; these include cardiac drift and fluid status.

3.2.1 Cardiovascular Drift

After the first few minutes of mild to moderate intensity exercise, there is a gradual decrease in stroke volume and increase in HR. This phenomenon of instability has been termed cardiac drift.[137,138] In a study published in 1967, Ekelund[138] described the cardiovascular changes which occurred in 18 individuals during 1 hour of exercise. HR was reported to increase gradually over the hour, with the largest increases during the first 30 minutes. HR had increased by 15% after 1 hour. Mognoni et al.[139] had individuals cycling at a constant work rate for 1 hour. HR increased from 135 beats/min after 10 minutes of exercise to 150 beats/min (11%) after 60 minutes.

It was speculated by Rowell[140] that the factors that are likely to contribute to the drift are a concomitant body water loss and a peripheral vasodilatation. Hamilton et al.[141] found that HR increased 10% when no fluid was consumed and 5% when fluid was provided to the individuals. It was concluded that half of the cardiovascular drift could be explained by dehydration. In the same study, it was shown that the rise in HR was closely related to the rise in body temperature.[141] It has been shown in several studies that when core temperature is increased, the HR showed a similar increase.[140,142-144]

When aiming for a certain HR zone during exercise, cardiac drift should be considered. Increases of up to 15% from 5–60 minutes of exercise have been reported. Cardiovascular drift is accentuated by numerous factors such as dehydration and heat stress. The effects of these factors on the cardiovascular system will be discussed in sections 3.2.2 and 3.3.1.

3.2.2 Hydration Status

The effects of dehydration on the cardiovascular system have been investigated extensively. In a study published in 1964, Saltin[145] dehydrated three individuals in a sauna. To investigate the effect of dehydration independent of hyperthermia, testing was only started after the core temperature had decreased to 37.2°C. Individuals lost between 1–5% bodyweight. A slight decrease in cardiac output was seen during submaximal exercise, with the magnitude of the decrease related to the decrease in bodyweight. This decrease in cardiac output was the

result of increased HR and a slightly larger relative decrease in stroke volume. Approximately 35 years later, in the late 1990s, Gonzàlez-Alonso et al.[142,143,146,147] investigated the effects of dehydration on the cardiovascular response to exercise. In a study published in 1997, seven endurance-trained athletes became 4% dehydrated or they remained euhydrated during a 100-minute cycle bout in the heat.[142] After a 45-minute rest, they cycled for 30 minutes at 70% $\dot{V}O_{2max}$ under cool conditions. HR and stroke volume changed significantly, with a 5% increase and 7% decrease, respectively, in the dehydrated athletes. When blood volume was restored to euhydrated levels, the stroke volume decline was reversed completely, despite the persistence of 3- to 4-litre extravascular dehydration. The authors concluded that the reduced stroke volume was induced by a reduced blood volume. Recently, a similar study was performed, looking at different levels of dehydration.[143] As was reported by Saltin,[145] both the changes in HR and stroke volume became larger when the body was more dehydrated. HRs were 2.5, 4.4 and 7.4% higher at 1.5, 3 and 4.2% dehydration, respectively.

When exercising in a dehydrated state, without a raised core temperature, HR can be increased up to 7.5%. The increase in HR is positively correlated to the level of dehydration. This means that when the body gets more dehydrated, using HR to monitor exercise intensity will become more and more unreliable.

3.3 Environmental Factors

Not only physiological factors can influence the relationship between HR and other exercise-related parameters. The environment will also have a large impact on HR. In this section the effects of ambient temperature and altitude will be described.

3.3.1 Temperature

Ambient temperature can have a large influence on the relationship between HR and $\dot{V}O_2$. Exercise tests in a laboratory are usually performed at 16–18°C. The HR-$\dot{V}O_2$ curve determined during such a test can only be accurately used to determine exercise intensity in the field when the ambient conditions are exactly the same. Both higher and lower temperatures can have relatively large influences on the interpretation of the intensity of an exercise bout. The influences of both hot and cool environments on the response to exercise have been extensively studied.

Heat

In almost all studies where exercise has been performed during hot conditions, it has been shown that HR increases.[142,143,147-151] For example, in a study by Gonzàlez-Alonso et al.,[143] individuals cycled for 30 minutes at 72% $\dot{V}O_{2max}$ either 8 or 35°C after they cycled for 2 hours at 60% $\dot{V}O_{2max}$ in 35°C. At the end of exercise the average HR was 150 beats/min in the cold environment and 160 beats/min in the hot environment ($p < 0.05$). Several factors have been suggested that could have influenced HR during hot conditions.

One possible factor influencing HR is core temperature. The measures of the body to lose heat (i.e. evaporation, convection, conduction, radiation) are only partially effective during hot conditions and eventually a rise in core temperature will occur. To investigate the relationship between HR and core temperature, Gonzàlez-Alonso et al.[149] performed a study where core temperature was manipulated prior to exercise in the heat. Seven male individuals performed three bouts of cycle ergometer exercise at 60% $\dot{V}O_{2max}$ at 40°C. Before exercise resting temperatures were altered by immersing individuals in water of 17, 36 or 40°C for 30 minutes. After 10 minutes of exercise it was shown that HR was 140 ± 5, 166 ± 5 and 182 ± 4 beats/min, respectively. It was concluded that HR increased gradually when the oesophageal temperature increased. Jose et al.[152] also reported a direct linear relationship between HR and mixed venous blood temperature. It

has also been suggested that HR during hot conditions can be increased by activation of muscle thermo-reflexes.[153]

Therefore, when exercise is performed in hot conditions, heat loss mechanisms are less efficient and core temperature increases. As a consequence, HR will be higher at the same exercise intensities. The increase in HR has been shown to be around 10 beats/min[143,149] and therefore overestimates the intensity of the exercise. While HR under these circumstances might not be the most accurate indicator of exercise intensity, it is a good marker of whole body stress.[83]

Cold

The two main adjustments that take place in a human when exposed to a cold environment are a decreased skin blood flow and an increased metabolic rate. Due to the increased temperature gradient between skin and environment, more heat will be lost through convection and radiation when remaining in cold temperatures.[84] To reduce the heat loss induced by these mechanisms, vasoconstriction of the skin blood vessels will occur and blood in the veins of the extremities is deviated from the superficial to the deep veins. This will increase both central blood volume and venous return.

Shivering is a reflex mechanism that the body uses to increase the metabolic rate. Since the mechanical efficiency of shivering is close to 0%, all the energy is being transferred into heat. Many muscle groups are involved in shivering, it can lead to a 2- to 4-fold increase in resting metabolic rate.[154] When resting, swimming or exercising on a cycle ergometer under water, $\dot{V}O_2$ has been shown to be higher when the temperature of the water is colder.[155-157] This increased $\dot{V}O_2$ is being used to cover the energy cost of shivering.

In 1968, Craig and Dvorak[155] performed a study with ten non-cold acclimatised individuals. The individuals either rested or performed cycle exercise at a low and high workload while being immersed in water with a temperature ranging from 24–37°C. A plateau in $\dot{V}O_2$ was seen from 37 to 32°C at rest, 37 to 28°C during low intensity exercise and 37–26°C in the high intensity trial. At the colder temperatures, marked increases in $\dot{V}O_2$ were seen. HR on the other hand remained low at the colder temperatures and increased when the water temperatures were increased. McArdle et al.[156] investigated the cardiovascular changes induced by a cold environment in more detail. Individuals exercised at six different exercise intensities in 26°C air, 18°C water or 25°C water. A significant increase in $\dot{V}O_2$ was seen in the 18 and 25°C water compared with the air trial. This difference became less pronounced during the higher exercise intensities. It was shown that the increased $\dot{V}O_2$ was most likely the result of an increased cardiac output. No differences were observed when HR was compared during the different temperature conditions at the same power output. An increase in stroke volume at lower temperatures caused the increase in cardiac output. It was speculated by the authors that this increased stroke volume was due to an increased central blood volume and venous return.[156]

During exercise in cold environments HR will be similar to that in thermoneutral conditions. However, $\dot{V}O_2$ will be higher and HR will therefore underestimate the intensity of exercise. During cool conditions, it is therefore often advised to athletes to train at the lower border of their training zones to obtain the required exercise intensity.

3.3.2 Altitude

Oxygen cost of work at altitude is essentially similar to the cost at sea level.[158,159] However, the partial pressure of ambient oxygen (PO_2) can be decreased to only 30% of the PO_2 at sea level at high altitudes (>4000m).[160] To compensate for this decrease in oxygen delivery per millilitre of blood, more blood needs to be shunted towards the exercising muscle. It has been shown that during submaximal exercise at altitude, the cardiac output will be

increased because of an increase in HR.[161] Vogel et al.[161] studied 16 men at sea level and after 2–3 days at 4300m. During mild exercise at altitude compared with sea level, HR increased 15%, and at moderate intensity exercise an increase of 10% was reported. Stenberg et al.[159] investigated six men at sea level and at 4000m simulated altitude. During mild and moderate exercise, increases in HR of 22 and 13% were found when altitude was compared with sea-level conditions. Klausen et al.[158] reported a 22% increase in HR during submaximal exercise at altitude compared with sea level. The nine individuals had an HR of 109 ± 41 beats/min at sea level and 141 ± 21 beats/min at altitude.

During maximal effort at altitude, however, HR has been shown to be the same or slightly reduced. The maximum HR of the individuals in the study of Vogel et al.[161] decreased from 180 to 176 beats/min after 2–3 days at altitude. In another study, maximum HR decreased from 186 to 184 beats/min at 4000m simulated altitude.[159] More marked reductions in maximal HR were found by Hartley and Saltin[162] HR_{max} decreased from 189 to 165 beats/min when maximal exercise was compared at 4600m altitude with sea level. Although HR has been shown to be increased at similar $\dot{V}O_2$, the relationship between $\dot{V}O_2$ and HR remains linear. The consequence is that at the same maximal HR, $\dot{V}O_2$ is reduced. Reductions in $\dot{V}O_{2max}$ of up to 70% have been reported.[159]

When the individuals' stay at altitude is longer than 3–4 days, some adjustments in the body take place. As a consequence, the cardiovascular responses to the reduced partial pressure change. Several studies have shown that submaximal cardiac output decreases and even returns to sea-level values.[161,163,164] This decrease is mainly caused by a decrease in stroke volume; HR remains elevated during the entire stay at altitude. After 12 days at 3800m, HR during submaximal exercise was 24 beats/min (18%) higher at altitude than at sea level.[158] Even after 3–4 weeks at 3800m, an elevation of almost 10% was observed.[163]

When exercising at altitude at a given $\dot{V}O_2$ the submaximal HR is increased while $\dot{V}O_2$ remains the same. A HR-$\dot{V}O_2$ curve determined at sea level will therefore not be suitable to use at altitude, since the exercise intensity will be overestimated.

4. Conclusions

Monitoring of HR has been used to evaluate responses to different exercise stressors for a long time. Between 1950 and 1980 a vast number of papers were published describing HR responses, and these continue to be well-cited today. Recently, the attention has shifted slightly towards the field of HRV. Given that low HRV is associated with increased mortality, it seems only logical that more research is pointed towards possible interventions to increase HRV. While age is negatively correlated to HRV, some evidence is arising indicating that training status and possibly exercise training can have a positive influence on HRV. In contrast to cross-sectional studies, which indicate that individuals with a higher $\dot{V}O_{2max}$ have higher HRV, longitudinal training studies have not been able to provide enough evidence to state that with exercise training, an increase in HRV can be achieved. Large controlled training studies using a variety of individuals should be performed to determine whether the actual training process can lead to increased HRV, or whether this is already genetically determined.

As becomes evident from section 2.2 on overtraining, there are still large gaps in our knowledge about the changes that occur during overtraining. The results from studies that have investigated the effects of overtraining on HR and HRV are mixed and more research is necessary to elucidate whether these parameters can be used to predict and therefore prevent overtraining.

Although technology has advanced quickly and it is possible to measure HR accurately and reliably

there is still little knowledge about the applications of HRMs. For instance, there is limited information about the exercise intensity required to provide the optimal training stimulus to improve performance. Most of this information remains anecdotal. Although several studies have been performed comparing steady-state exercise with intermittent exercise, as well as studies looking at intermittent programmes using different intensities and different duration, no clear guidelines exist for the optimal training stimulus to obtain various training adaptations.

So even though the topic of HR and HR monitoring has received a vast amount of attention in the literature over the last 50–60 years, there are still areas in the HR monitoring field that need elucidation.

Acknowledgements

The authors would like to thank Dr Raija Laukannen, Hannu Kinnunen and Dr Mike White for their careful and critical reviewing of this manuscript. No sources of funding have been used in the preparation of this manuscript and there are no conflicts of interest directly relevant to the content of this review.

References

1. Holter NJ. New method for heart studies. Science 1961; 134: 1214-20
2. Laukkanen RMT, Virtanen PK. Heart rate monitors: state of the art. J Sport Sci 1998; 16: S3-7
3. Tsuji H, Venditti Jr FJ, Manders ES, et al. Reduced heart rate variability and mortality risk in an elderly cohort: the Framingham Heart Study. Circulation 1994; 90 (2): 878-83
4. Tsuji H, Larson MG, Venditti Jr FJ, et al. Impact of reduced heart rate variability on risk for cardiac events: the Framingham Heart Study. Circulation 1996; 94 (11): 2850-5
5. Molgaard H, Sorensen KE, Bjerregaard P. Attenuated 24-h heart rate variability in apparently healthy subjects, subsequently suffering sudden cardiac death. Clin Auton Res 1991; 1 (3): 233-7
6. Akselrod S, Gordon D, Ubel FA, et al. Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. Science 1981; 213 (4504): 220-2
7. Hayano J, Sakakibara Y, Yamada A, et al. Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. Am J Cardiol 1991; 67 (2): 199-204
8. Kamath MV, Fallen EL. Power spectral analysis of heart rate variability: a noninvasive signature of cardiac autonomic function. Crit Rev Biomed Eng 1993; 21 (3): 245-311
9. Malliani A, Pagani M, Lombardi F, et al. Cardiovascular neural regulation explored in the frequency domain. Circulation 1991; 84 (2): 482-92
10. Rimoldi O, Pierini S, Ferrari A, et al. Analysis of short-term oscillations of R-R and arterial pressure in conscious dogs. Am J Physiol 1990; 258 (4 Pt 2): H967-76
11. Pomeranz B, Macaulay RJ, Caudill MA, et al. Assessment of autonomic function in humans by heart rate spectral analysis. Am J Physiol 1985; 248 (1 Pt 2): H151-3
12. Eckberg DL. Human sinus arrythmia as an index of vagal cardiac outflow. J Appl Physiol 1983; 54: 961-6
13. Pichot V, Roche F, Gaspoz J-M, et al. Relation between heart rate variability and training load in middle distance runners. Med Sci Sport Exerc 2000; 32 (10): 1729-36
14. Nakamura Y, Yamamoto Y, Muraoka I. Autonomic control of heart rate during physical exercise and fractal dimension of heart rate variability. J Appl Physiol 1993; 74 (2): 875-81
15. Melanson EL. Resting heart rate variability in men varying in habitual physical activity. Med Sci Sports Exerc 2000; 32 (11): 1894-901
16. Kleiger RE, Bigger JT, Bosner MS. Stability over time of variables measuring heart rate variability in normal subjects. Am J Physiol 1997; 68: 626-30
17. Jensen-Urstad K, Storck N, Bouvier F, et al. Heart rate variability in healthy subjects is related to age and gender. Acta Physiol Scand 1997; 160 (3): 235-41
18. Liao D, Barnes RW, Chambless LE, et al. Age, race, and sex differences in autonomic cardiac function measured by spectral analysis of heart rate variability: the ARIC study. Atherosclerosis risk in communities. Am J Cardiol 1995; 76 (12): 906-12
19. Bigger Jr JT, Fleiss JL, Steinman RC, et al. RR variability in healthy, middle-aged persons compared with patients with chronic coronary heart disease or recent acute myocardial infarction. Circulation 1995; 91 (7): 1936-43
20. Schwartz JB, Gibb WJ, Tran T. Aging effects on heart rate variation. J Gerontol 1991; 46 (3): M99-106
21. Ziegler D, Laux G, Dannehl K, et al. Assessment of cardiovascular autonomic function: age-related normal ranges and reproducibility of spectral analysis, vector analysis, and standard tests of heart rate variation and blood pressure responses. Diabet Med 1992; 9 (2): 166-75
22. Brown TE, Beightol LA, Koh J, et al. Important influence of respiration on human R-R interval power spectra is largely ignored. J Appl Physiol 1993; 75 (5): 2310-7
23. Pagani M, Lombardi F, Guzzetti S, et al. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circ Res 1986; 59 (2): 178-93
24. Perini R, Orizio C, Milesi S, et al. Body position affects the power spectrum of heart rate variability during dynamic exercise. Eur J Appl Physiol 1993; 66 (3): 207-13
25. O'Leary DS, Seamans DP. Effect of exercise on autonomic mechanisms of baroreflex control of heart rate. J Appl Physiol 1993; 75 (5): 2251-7

26. Brenner IKM, Thomas S, Shephard RJ. Autonomic regulation of the circulation during exercise and heat exposure. Sports Med 1998; 26 (2): 85-99
27. Casadei B, Cochrane S, Johnston J, et al. Pitfalls in the interpretation of spectral analysis of the heart rate variability during exercise in humans. Acta Physiol Scand 1995; 153 (2): 125-31
28. Dixon EM, Kamath MV, McCartney N, et al. Neural regulation of heart rate variability in endurance athletes and sedentary controls. Cardiovasc Res 1992; 26 (7): 713-9
29. Gregoire J, Tuck S, Yamamoto Y, et al. Heart rate variability at rest and exercise: influence of age, gender, and physical training. Can J Appl Physiol 1996; 21 (6): 455-70
30. Yamamoto Y, Hughson RL, Peterson JC. Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis. J Appl Physiol 1991; 71 (3): 1136-42
31. Amara CE, Wolfe LA. Spectral analysis of heart rate variability (HRV) at rest and during exercise: reliability and reproducibility. Med Sci Sports Exerc 1997; 29: S24
32. Perini R, Orizio C, Gamba A, et al. Kinetics of heart rate and catecholamines during exercise in humans: the effect of heart denervation. Eur J Appl Physiol 1993; 66 (6): 500-6
33. Arai Y, Saul JP, Albrecht P, et al. Modulation of cardiac autonomic activity during and immediately after exercise. Am J Physiol 1989; 256 (1 Pt 2): H132-41
34. Breuer HW, Skyschally A, Schulz R, et al. Heart rate variability and circulating catecholamine concentrations during steady state exercise in healthy volunteers. Br Heart J 1993; 70 (2): 144-9
35. Gonzalez-Camarena R, Carrasco-Sosa S, Roman-Ramos R, et al. Effect of static and dynamic exercise on heart rate and blood pressure variabilities. Med Sci Sports Exerc 2000; 32 (10): 1719-28
36. Dickhuth HH, Lehmann M, Auch-Schwelk W, et al. Physical training, vegetative regulation, and cardiac hypertrophy. J Cardiovasc Pharmacol 1987; 10 Suppl. 6: S71-8
37. Katona PG, McLean M, Dighton DH, et al. Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. J Appl Physiol 1982; 52 (6): 1652-7
38. Lewis SF, Nylander E, Gad P, et al. Non-autonomic component in bradycardia of endurance trained men at rest and during exercise. Acta Physiol Scand 1980; 109 (3): 297-305
39. Bedford TG, Tipton CM. Exercise training and the arterial baroreflex. J Appl Physiol 1987; 63 (5): 1926-32
40. de Schryver C, Mertens-Strythagen J. Heart tissue acetylcholine in chronically exercised rats. Experientia 1975; 31 (3): 316-8
41. Ekblom B, Kilbom A, Soltysiak J. Physical training, bradycardia, and autonomic nervous system. Scand J Clin Lab Invest 1973; 32 (3): 251-6
42. Fraser J, Nadeau J, Robertson D, et al. Regulation of human leukocyte beta receptors by endogenous catecholamines: relationship of leukocyte beta receptor density to the cardiac sensitivity to isoproterenol. J Clin Invest 1981; 67 (6): 1777-84
43. Stegemann J, Busert A, Brock D. Influence of fitness on the blood pressure control system in man. Aerosp Med 1974; 45 (1): 45-8
44. Butler J, O'Brien M, O'Malley K, et al. Relationship of beta-adrenoreceptor density to fitness in athletes. Nature 1982; 298 (5869): 60-2
45. Cousineau D, Ferguson RJ, de Champlain J, et al. Catecholamines in coronary sinus during exercise in man before and after training. J Appl Physiol 1977; 43 (5): 801-6
46. Lehmann M, Keul J, Berg A, et al. Plasma catecholamines and aerobic-anaerobic capacity in women during graduated treadmill exercise [in German]. Eur J Appl Physiol Occup Physiol 1981; 46 (3): 305-15
47. Vatner DE, Lavallee M, Amano J, et al. Mechanisms of supersensitivity to sympathomimetic amines in the chronically denervated heart of the conscious dog. Circ Res 1985; 57 (1): 55-64
48. Bonaduce D, Petretta M, Cavallaro V, et al. Intensive training and cardiac autonomic control in high level athletes. Med Sci Sports Exerc 1998; 30 (5): 691-6
49. Davy KP, Miniclier NL, Taylor JA, et al. Elevated heart rate variability in physically active postmenopausal women: a cardioprotective effect? Am J Physiol 1996; 271 (2 Pt 2): H455-60
50. Goldsmith RL, Bigger JT, Bloomfield DM, et al. Physical fitness as a determinant of vagal modulation. Med Sci Sports Exerc 1997; 29 (6): 812-7
51. Grund A, Krause H, Kraus M, et al. Association between different attributes of physical activity and fat mass in untrained, endurance- and resistance-trained men. Eur J Appl Physiol 2001; 84 (4): 310-20
52. Janssen MJ, de Bie J, Swenne CA, et al. Supine and standing sympathovagal balance in athletes and controls. Eur J Appl Physiol 1993; 67 (2): 164-7
53. Macor F, Fagard R, Amery A. Power spectral analysis of RR interval and blood pressure short-term variability at rest and during dynamic exercise: comparison between cyclists and controls. Int J Sports Med 1996; 17 (3): 175-81
54. Puig J, Freitas J, Carvalho MJ, et al. Spectral analysis of heart rate variability in athletes. J Sports Med Phys Fitness 1993; 33 (1): 44-8
55. Sacknoff DM, Gleim GW, Stachenfeld N, et al. Effect of athletic training on heart rate variability. Am Heart J 1994; 127 (5): 1275-8
56. Yataco AR, Fleisher LA, Katzel LI. Heart rate variability and cardiovascular fitness in senior athletes. Am J Cardiol 1997; 80 (10): 1389-91
57. Hedelin R, Bjerle P, Henriksson-Larsen K. Heart rate variability in athletes: relationship with central and peripheral performance. Med Sci Sports Exerc 2001; 33 (8): 1394-8
58. Shin K, Minamitani H, Onishi S, et al. Autonomic differences between athletes and nonathletes: spectral analysis approach. Med Sci Sports Med 1997; 29 (11): 1482-90
59. Levy WC, Cerqueira MD, Harp GD, et al. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. Am J Cardiol 1998; 82 (10): 1236-41
60. Schuit AJ, van Amelsvoort LG, Verheij TC, et al. Exercise training and heart rate variability in older people. Med Sci Sports Exerc 1999; 31 (6): 816-21

61. Seals DR, Chase PB. Influence of physical training on heart rate variability and baroreflex circulatory control. J Appl Physiol 1989; 66 (4): 1886-95
62. Stein PK, Ehsani AA, Domitrovich PP, et al. Effect of exercise training on heart rate variability in healthy older adults. Am Heart J 1999; 138 (3 Pt 1): 567-76
63. al-Ani M, Munir SM, White M, et al. Changes in R-R variability before and after endurance training measured by power spectral analysis and by the effect of isometric muscle contraction. Eur J Appl Physiol 1996; 74 (5): 397-403
64. Amano M, Kanda T, Ue H, et al. Exercise training and autonomic nervous system activity in obese individuals. Med Sci Sports Exerc 2001; 33 (8): 1287-91
65. Melanson EL, Freedson PS. The effect of endurance training on resting heart rate variability in sedentary adult males. Eur J Appl Physiol 2001; 85 (5): 442-9
66. Portier H, Louisy F, Laude D, et al. Intense endurance training on heart rate and blood pressure variability in runners. Med Sci Sports Exerc 2001; 33 (7): 1120-5
67. Boutcher SH, Stein P. Association between heart rate variability and training response in sedentary middle-aged men. Eur J Appl Physiol Occup Physiol 1995; 70 (1): 75-80
68. Loimaala A, Huikuri H, Oja P, et al. Controlled 5-mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. J Appl Physiol 2000; 89 (5): 1825-9
69. Perini R, Fisher N, Veicsteinas A, et al. Aerobic training and cardiovascular responses at rest and during exercise in older men and women. Med Sci Sports Exerc 2002; 34 (4): 700-8
70. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Circulation 1996; 93 (5): 1043-65
71. Léger L, Thivierge M. Heart rate monitors: validity, stability and functionality. Physician Sports Med 1988; 16 (5): 143-51
72. Thivierge M, Leger L. Validité des cardiofréquencemètres. Sci Sports 1988; 3: 211-221.
73. Macfarlane DJ, Fogarty B, Hopkins WG. The accuracy and variability of commercially available heart rate monitors. N Z J Sports Med 1989; 17 (4): 51-3
74. Seaward B, Sleamakee R, McAuliffe T, et al. The precision and accuracy of a portable heart rate monitor. Biomed Instrument Technol 1990; 24: 37-41
75. Godsen R, Carroll T, Stone S. How well does Polar Vantage XL heart rate monitor estimate actual heart rate? Med Sci Sports Exerc 1991; 23 Suppl. 5: S14
76. Goodie JL, Larkin KT, Schauss S. Validation of the Polar heart rate monitor for assessing heart rate during physical and mental stress. J Psychophysiol 2000; 14 (3): 159-64
77. Kinnunen H, Heikkila I. The timing accuracy of the Polar Vantage NV heart rate monitor. J Sports Sci 1998; 16: S107-10
78. Ruha A, Salinnen S, Nissala S. A real-time microprocessor QRS detector system with a 1 ms timing accuracy for the measurement of ambulatory HRV. IEEE Trans Biomed Eng 1997; 44: 159-67
79. Bannister E. Modeling elite athletic performance. In: Green H, McDougall J, Wenger H, editors. Physiological testing of elite athletes. Champaign (IL): Human Kinetics, 1991: 403-24
80. Jeukendrup AE, Hesselink MKC, Kuipers H, et al. Physiological changes in male competitive cyclists after two weeks of intensified training. Int J Sports Med 1992; 13 (7): 534-41
81. Lehmann M, Baumgartl P, Wiesenack C, et al. Training-overtraining: influence of a defined increase in training volumes vs training intensity on performance, catecholamines and some metabolic parameters in experienced middle- and long-distance runners. Eur J Appl Physiol 1992; 64 (2): 169-77
82. Lehmann M, Dickhuth HH, Gendrisch G, et al. Training-overtraining: a prospective, experimental study with experienced middle- and long-distance runners. Int J Sports Med 1991; 12 (5): 444-52
83. Jeukendrup A, Van Diemen A. Heart rate monitoring during training and competition in cyclists. J Sports Sci 1998; 16: 1-9
84. Astrand P, Rodahl K. Textbook of work physiology. New York: McGraw Hill, 1986
85. American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. Med Sci Sports Exerc 1998; 30 (6): 975-91
86. Arkinstall MJ, Bruce CR, Nikolopoulos V, et al. Effect of carbohydrate ingestion on metabolism during running and cycling. J Appl Physiol 2001; 91 (5): 2125-34
87. Padilla S, Mujika I, Orbananos J. Exercise intensity and load during mass-start stage races in professional road cycling. Med Sci Sport Exerc 2001; 33 (5): 796-802
88. Halson SL, Bridge MW, Meeuwsen R, et al. Time course of performance changes and fatigue markers during intensified training in trained cyclists. J Appl Physiol 2002; 93 (3): 947-56
89. Conconi F, Ferrari M, Ziglio PG, et al. Determination of the anaerobic threshold by a noninvasive field test in runners. J Appl Physiol 1982; 52 (4): 869-73
90. Bodner ME, Rhodes EC. A review of the concept of the heart rate deflection point. Sports Med 2000; 30 (1): 31-46
91. Kuipers H, Keizer HA, de Vries T, et al. Comparison of heart rate as a non-invasive determinant of anaerobic threshold with the lactate threshold when cycling. Eur J Appl Physiol 1988; 58 (3): 303-6
92. Leger L, Tokmakidis S. Use of the heart rate deflection point to assess the anaerobic threshold. J Appl Physiol 1988; 64 (4): 1758-60
93. Tokmakidis SP, Leger LA. Comparison of mathematically determined blood lactate and heart rate 'threshold' points and relationship with performance. Eur J Appl Physiol 1992; 64 (4): 309-17
94. Jeukendrup AE, Hesselink MK, Kuipers H, et al. The Conconi test. Int J Sports Med 1997; 18 (5): 393-6
95. Conconi F, Grazzi G, Casoni I, et al. The Conconi test: methodology after 12 years of application. Int J Sports Med 1996; 17 (7): 509-19
96. Vachon JA, Bassett Jr DR, Clarke S. Validity of the heart rate deflection point as a predictor of lactate threshold during running. J Appl Physiol 1999; 87 (1): 452-9
97. Hedelin R, Kentta G, Wiklund U, et al. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. Med Sci Sports Exerc 2000; 32 (8): 1480-4

98. Urhausen A, Gabriel HH, Weiler B, et al. Ergometric and psychological findings during overtraining: a long-term follow-up study in endurance athletes. Int J Sports Med 1998; 19 (2): 114-20
99. Uusitalo AL, Uusitalo AJ, Rusko HK. Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. Int J Sports Med 2000; 21 (1): 45-53
100. Israel S. Die erscheinungsformen des ubertrainings. Med Sport 1958; 9: 207-9
101. Snyder AC, Kuipers H, Cheng B, et al. Overtraining following intensified training with normal muscle glycogen. Med Sci Sports Exerc 1995; 27 (7): 1063-70
102. Dressendorfer RH, Wade CE, Schaff JH. Increased heart rate in runners: a valid sign of overtraining? Physician Sports Med 1985; 13: 77-86
103. Kindermann W. Overtraining: expression of a disturbed autonomic regulation [in German]. Dtsch Z Sportmed 1986; 37: 238-45
104. Stray-Gundersen J, Videman T, Snell PG. Changes in selected parameters during overtraining. Med Sci Sports Exerc 1986; 18: S54-5
105. Billat VL, Flechet B, Petit B, et al. Interval training at V̇O2max: effects on aerobic performance and overtraining markers. Med Sci Sports Exerc 1999; 31 (1): 156-63
106. Costill DL, Flynn MG, Kirwan JP, et al. Effects of repeated days of intensified training on muscle glycogen and swimming performance. Med Sci Sports Exerc 1988; 20 (3): 249-54
107. Urhausen A, Gabriel HH, Kindermann W. Impaired pituitary hormonal response to exhaustive exercise in overtrained endurance athletes. Med Sci Sports Exerc 1998; 30 (3): 407-14
108. Hedelin R, Wiklund U, Bjerle P, et al. Cardiac autonomic imbalance in an overtrained athlete. Med Sci Sports Exerc 2000; 32 (9): 1531-3
109. Furlan R, Piazza S, Dell'Orto S, et al. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. Cardiovasc Res 1993; 27 (3): 482-8
110. McArdle W, Katch F, Katch V. Exercise physiology: energy, nutrition and human performance. 4th ed. Baltimore (MD): Williams and Wilkins, 1996
111. Andrew GM, Guzman CA, Becklake MR. Effect of athletic training on exercise cardiac output. J Appl Physiol 1966; 21 (2): 603-8
112. Clausen JP, Trap-Jensen J, Lassen NA. The effects of training on the heart rate during arm and leg exercise. Scand J Clin Invest 1970; 26: 295-301
113. Cotes JE, Meade F. Physical training in relation to the energy expenditure of walking and to factors controlling respiration. Ergonomics 1959; 2: 195-206
114. Ekblom B, Astrand P-O, Saltin B, et al. Effect of training on circulatory response to exercise. J Appl Physiol 1968; 24 (4): 518-28
115. Londeree BR, Ames SA. Trend analysis of the %V̇O2max-HR regression. Med Sci Sports Exerc 1976; 8 (2): 122-5
116. Astrand P-O, Ryhming I. Nomogram for calculations of aerobic capacity from pulse rate during submaximal work [abstract]. J Appl Physiol 1954; 7: 218
117. Davies CT. Limitations to the prediction of maximum oxygen intake from cardiac frequency measurements. J Appl Physiol 1968; 24 (5): 700-6
118. Miller WC, Wallace JP, Eggert KE. Predicting max HR and the HR-VO2 relationship for exercise prescription in obesity. Med Sci Sports Exerc 1993; 25 (9): 1077-81
119. Whaley MH, Kaminsky LA, Dwyer GB, et al. Predictors of over- and underachievement of age-predicted maximal heart rate. Med Sci Sports Exerc 1992; 24 (10): 1173-9
120. Kinnunen H, Vainamo K, Hautala A, et al. Artificial neural network in predicting maximal aerobic power. Med Sci Sport Exerc 2000; 32 (5 Suppl.): S308
121. Laukkanen R. Scientific development and evaluation of the Polar Fitness test™ [online]. Available from URL: http://www.polarkorea.co.kr/resech/SCIENTIFICDEVELOPMENT.htm [Accessed 2003 May 5]
122. Spurr GB, Prentice AM, Murgatroyd PR, et al. Energy expenditure from minute-by-minute heart-rate recording: comparison with indirect calorimetry. Am J Clin Nutr 1988; 48: 552-9
123. Andrews RB. Net heart rate as a substitute for respiratory calorimetry. Am J Clin Nutr 1971; 24: 1139-47
124. Dauncey MJ, James WPT. Assessment of heart rate method for determining energy expenditure in man, using a whole-body calorimeter. Br J Nutr 1979; 42: 1-13
125. Davidson L, McNeill G, Haggarty P, et al. Free-living energy expenditure of adult men assessed by continuous heart rate monitoring and doubly-labelled water. Br J Nutr 1997; 79: 695-708
126. Li R, Deurenberg P, Hautvast JGAJ. A critical evaluation of heart rate monitoring to assess energy expenditure in individuals. Am J Clin Nutr 1993; 58: 602-7
127. Luke A, Maki KC, Barkey N, et al. Simultaneous monitoring of heart rate and motion to assess energy expenditure. Med Sci Sports Exerc 1997; 29 (1): 144-8
128. Treuth MS, Adolph AL, Butte NF. Energy expenditure in children predicted fom heart rate and activity against respiration calorimetry. Am J Physiol 1998; 275: E12-8
129. Andrews RB. Indices of heart rate as substitutes for respiratory calorimetry. Am Ind Hyg Assoc J 1966; 27 (6): 526-32
130. Ceesay SM, Prentice AM, Day KC, et al. The use of heart rate monitoring in the estimation of energy expenditure: a validation study using indirect whole-body calorimetry. Br J Nutr 1989; 61: 175-86
131. McCrory MA, Mole PA, Nommsen-Rivers LA, et al. Between-day and within-day variation in the relation between heart rate and oxygen consumption: effect on the estimation of energy expenditure by heart-rate monitoring. Am J Clin Nutr 1997; 66: 18-25
132. Taylor C. Some properties of maximal and submaximal exercise with reference to physiological variation and the measurement of exercise tolerance. Am J Physiol 1944; 142: 200-12
133. Astrand PO, Saltin B. Oxygen uptake during the first minutes of heavy muscular exercise. J Appl Physiol 1961; 16 (6): 971-6
134. Brooke JD, Hamley EJ, Thomason H. Variability in the measurement of exercise heart rate. J Sports Med Phys Fitness 1970; 10 (1): 21-5
135. Becque MD, Katch V, Marks C, et al. Reliability and within subject variability of VE, VO2, heart rate and blood pressure

during maximum cycle ergometry. Int J Sports Med 1993; 14 (4): 220-3

136. Brisswalter J, Legros P. Daily stability in energy cost of running, respiratory parameters and stride rate among well-trained middle distance runners. Int J Sports Med 1994; 15 (5): 238-41

137. Coyle EF. Cardiovascular drift during prolonged exercise and the effects of dehydration. Int J Sports Med 1998; 19 Suppl. 2: S121-4

138. Ekelund LG. Circulatory and respiratory adaptations during prolonged exercise. Acta Physiol Scand 1967; 70 Suppl. 68: 5-38

139. Mognoni P, Sirtoni MD, Lorenzelli F, et al. Physiological responses during prolonged exercise at the power output corresponding to the blood lactate threshold. Eur J Appl Physiol 1990; 60: 239-43

140. Rowell LB. Human cardiovascular adjustment to exercise and thermal stress. Physiol Rev 1974; 54: 75-159

141. Hamilton MT, Gonzàlez-Alonso J, Montain SJ, et al. Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. J Appl Physiol 1991; 71 (3): 871-7

142. Gonzàlez-Alonso J, Mora-Rodriguez R, Below PR, et al. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. J Appl Physiol 1997; 82 (4): 1229-36

143. Gonzàlez-Alonso J, Mora-Rodriguez R, Coyle EF. Stroke volume during exercise: interaction of environment and hydration. Am J Physiol Heart Circ Physiol 2000; 278: H321-30

144. Gonzàlez-Alonso J, Mora-Rodriguez R, Coyle EF. Supine exercise restores arterial blood pressure and skin blood flow despite dehydration and hyperthermia. Am J Physiol 1999; 277: H576-83

145. Saltin B. Circulatory response to submaximal and maximal exercise after thermal dehydration. J Appl Physiol 1964; 19 (6): 1125-32

146. Gonzàlez-Alonso J. Separate and combined influences of dehydration and hyperthermia on cardiovascular responses to exercise. Int J Sports Med 1998; 19: S111-4

147. Gonzàlez-Alonso J, Mora-Rodriguez R, Below PR, et al. Dehydration reduces cardiac output and incerases systemic and cutaneous vascular resistance during exercise. J Appl Physiol 1995; 79: 1487-96

148. Fritzsche RG, Switzer TW, Hodginkinson BJ, et al. Stroke volume decline during prolonged exercsie is influences by the increase in heart rate. J Appl Physiol 1999; 86 (3): 799-805

149. Gonzàlez-Alonso J, Teller C, Andersen SL, et al. Influence of body temperature in the development of fatigue during prolonged exercise in the heat. J Appl Physiol 1999; 86 (3): 1032-9

150. Rowell LB, Marx HJ, Bruce RA, et al. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J Clin Invest 1966; 45: 1801-16

151. Rowell LB, Brengelmann GL, Freund PR. Unaltered norepinephrine: heart rate relationship in exercise with exogenous heat. J Appl Physiol 1987; 62: 646-50

152. Jose AD, Stitt F, Collison D. The effects of exercise and changes in body temperature on the intrinsic heart rate in man. Am Heart J 1970; 79 (4): 488-98

153. Rowell LB, O'Leary DS, Kellog Jr DL. Integration of cardiovascular control systems in dynamic exercise. In: Rowell LB, Sheppard JT, editors. Handbook of physiology. Exercise: regulation and integration of multiple systems. New York: Oxford University Press, 1996: 770-838

154. Bell DP, Tikuisis P, Jacobs I. Relative intensity of muscular contraction during shivering. J Appl Physiol 1992; 72: 2336-42

155. Craig AB, Dvorak M. Thermal regulation of man exercising during water immersion. J Appl Phsyiol 1968; 25 (1): 28-35

156. McArdle WD, Magel JR, Lesmes GR, et al. Metabolic and cardiovascular adjustment to work in air and water at 18, 25 and 33 degrees C. J Appl Physiol 1976; 40 (1): 85-90

157. McArdle WD, Magel JR, Spina RJ, et al. Thermal adjustment to cold-water exposure in exercising men and women. J Appl Physiol 1984; 56 (6): 1572-7

158. Klausen K, Dill DB, Horvath SM. Exercise at ambient and high oxygen pressure at high altitude and at sea level. J Appl Physiol 1970; 29 (4): 456-63

159. Stenberg J, Ekblom B, Messin R. Hemodynamic response to work at simulated altitude, 4.000m. J Appl Physiol 1966; 21 (5): 1589-94

160. West JB. Do climbs to extreme altitudes cause brain damage? Lancet 1986; II: 387-8

161. Vogel JA, Hansen JE, Harris CW. Cardiovascular responses in man during exhaustive work at sea level and high altitude. J Appl Physiol 1967; 23 (4): 531-9

162. Hartley LH, Saltin B. Reduction of stroke volume and increase in heart rate after a previous heavier submaximal work load. Scand J Clin Invest 1968; 22: 217-23

163. Klausen K. Cardiac output in man in rest and work during and after acclimatization to 3,800m. J Appl Physiol 1966; 21 (2): 609-16

164. Pugh LGCE. Cardiac output in muscular exercise at 5800m. J Appl Physiol 1964; 19: 441-7

Correspondence and offprints: *Asker E. Jeukendrup*, Human Performance Laboratory, School of Sport and Exercise Sciences, University of Birmingham, Edgbaston, Birmingham, B15 2TT, United Kingdom.
E-mail: A.E.Jeukendrup@bham.ac.uk